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Hazards of hypoxemia: How to protect your patient from low oxygen levels

Nursing CE Handbook March 1998

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Hypoxemia can threaten almost any patient, complicating his recovery. Learn to spot problems early and keep your patient safe.

Richard Shore, an obese 42-year-old patient in your surgical unit, is in his second postoperative day after abdominal aortic aneurysm repair. He's receiving morphine at about 2 to 4 mg/hour via his patient-controlled analgesia pump. He has no preexisting medical conditions and his postoperative course has been uncomplicated, but he's somewhat sluggish.

When assessing Mr. Shore as you begin your night shift, you start with a visual and vital signs assessment. He appears drowsy and slightly confused. His vital signs include a temperature of 98.6° F (37° C); heart rate, 94; respirations, 12; and blood pressure, 144/92. Mr. Shore's color is slightly pale (as it's been since his surgery) and his abdominal dressing is dry and intact. He's had 16 mg of morphine during the last 6 hours. Should you attribute the drowsiness and slight confusion to a combination of morphine, the late hour, and Mr. Shore's lack of sleep?

Possibly--but don't jump to that conclusion too quickly. Mr. Shore has several risk factors that make him a likely candidate for hypoxemia, which is now recognized as a common complication in many settings outside of traditional critical care.

Using the call light, you ask a nursing assistant to bring in a portable pulse oximeter. When you place the sensor on Mr. Shore's index finger, the device gives a reading of 89%, well below the normal range of 95% to 100%. You immediately initiate oxygen via nasal cannula at 4 liters/ minute and notify Mr. Shore's physician. He orders arterial blood gases (ABGs), continuous pulse oximetry monitoring, and a chest X-ray to rule out atelectasis or pulmonary infection.

Because hypoxemia can lead to tissue damage, you need to learn as much as possible about it to identify high-risk patients. You also need to routinely monitor your patients for signs of hypoxemia and implement a plan of nursing actions when appropriate (see *Preventing Problems*). Let's start by specifying what hypoxemia means.

Defining our terms

In general, *hypoxemia* refers to a lowered oxygen level in the blood. Some definitions specify that the partial pressure of arterial oxygen (PaO_2) is less than 60 mm Hg; other standards set the arterial oxygen saturation value (SaO_2) at less than 90%. The *oxygen saturation* level refers to the percentage of hemoglobin that's saturated with oxygen.

In the lungs, oxygen diffuses across the alveolar-capillary membrane into arterial blood. Ninety-eight percent to 99% of oxygen is combined with hemoglobin in the red blood cell to form oxyhemoglobin. The rest (1% to 2%) is dissolved in plasma, where it exerts a gas pressure, PaO_2.

Oxyhemoglobin is carried to the tissues, where the pressure of oxygen is lower. Oxygen moves from the hemoglobin to the plasma, while oxygen simultaneously moves from the plasma to the body cells. When tissues receive too little oxygen, *hypoxia* (low tissue oxygenation) may result, causing tissue damage and even cell death.

Several key causes

Hypoxemia's many causes can be categorized as follows:

- *Low levels of inspired oxygen* can occur at extremely high altitudes where partial pressures of oxygen are lower or when room air contains less oxygen than the patient requires.
- *Inadequate lung ventilation* can have many causes. For example, ventilatory drive may be compromised by neurologic

disease or the depressant effects of anesthesia or narcotics. Musculoskeletal problems may handicap respiratory muscles. And airway obstruction, pneumonia, and atelectasis limit the amount of alveoli available for ventilation.

- *Impaired oxygen diffusion* occurs when the alveolar-capillary membrane thickens, limiting gas diffusion. Clinical conditions that can thicken membranes include chronic bronchitis, acute respiratory distress syndrome, fibrosis, and pulmonary edema.

- *Diminished levels of pulmonary circulation* can be caused by perfusion abnormalities. For example, a pulmonary embolus limits the size of the pulmonary capillary bed available for diffusion.

- *Lowered blood oxygen* levels can result from anemia, carbon monoxide poisoning, and methemoglobinemia.

Getting oxygen to its goal

For your patient to be adequately oxygenated, three conditions must be met: Blood oxygen levels must be sufficient, oxygen must be delivered efficiently to tissues, and cells must be able to extract and use the oxygen to meet metabolic needs. Let's look more closely at each level of oxygenation.

- *Ensuring adequate blood oxygen levels* is the first step in the oxygenation process. The key ingredients are adequate inspired oxygen, efficient diffusion of oxygen across the alveolar-capillary membrane into arterial blood, and sufficient amounts of hemoglobin molecules. Reduced or inadequate inspired oxygen, impaired oxygen diffusion, and lowered hemoglobin levels--separately or in combination--can impair blood oxygen levels and lower total oxygen content in the body.

- *Efficient delivery* of oxygen to the tissues requires adequate cardiac output and unencumbered perfusion to the tissues. Reduced blood flow or impaired local or systemic perfusion hampers oxygen delivery to the tissues.

- Finally, the ability of cells to *extract and use* oxygen requires adequate cellular uptake. Abnormal cellular processes can alter cellular oxygen use. Hypoglycemia, for example, can impair a cell's ability to process oxygen and causes hypoxia.

These factors come into play with both subacute and episodic hypoxemia. In *subacute* (chronic) hypoxemia, oxygen saturation falls to a consistently lower but noncritical level. A patient with subacute hypoxemia (for example, someone with chronic obstructive pulmonary disease) may breathe normally. However, the combination of subacute hypoxemia with inadequate oxygen delivery or increased metabolic demand may spell trouble for him.

With *episodic* hypoxemia, oxygen saturation may fall quickly and

BLOOD GAS QUESTIONS WITH ANSWERS - 1998

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This Quiz ran from February through December, 1998. It is based on Dr. Martin's ***All You Really Need to Know to Interpret Arterial Blood Gases***, 2nd edition, published February 1999 by Lippincott Williams & Wilkins. Click on the title to see the Preface and Table of Contents. The book is available for purchase from the publisher at 1-800-638-0672 or 1-410-528-4223 and at the following web sites:

Lippincott Williams & Wilkins
Amazon.com

In these questions, the degree of difficulty ranges from 1 -5; (1 is easiest, 5 most difficult.)

QUESTION FOR FEBRUARY 12, 1998 (degree of difficulty 2/5)

Which patient is more hypoxemic, and why?

Patient A: pH 7.48, $PaCO_2$ 34 mm Hg, PaO_2 85 mm Hg, SaO_2 95%, Hemoglobin 7 gm%

Patient B: pH 7.32, $PaCO_2$ 74 mm Hg, PaO_2 55 mm Hg, SaO_2 85%, Hemoglobin 15 gm%

Hint: Be specific -- this is not a question you guess at.

ANSWER TO FEBRUARY 12 QUESTION

The body needs oxygen molecules, so oxygen content takes precedence over partial pressure in determining degree of hypoxemia. In this problem the amount of oxygen molecules contributed by the dissolved fraction is negligible and will not affect the answer. Also, the PaCO2 and pH are not needed to answer the question.

Patient A: Arterial oxygen content = .95 x 7 x 1.34 = 8.9 ml O2/dl

Patient B: Arterial oxygen content = .85 x 15 x 1.34 = 17.1 ml O2/dl

Patient A, with the higher PaO2 but the lower hemoglobin content, is more hypoxemic.

Reference: **O2 Content Equation**

QUESTION FOR FEBRUARY 18, 1998

True or False:

PaO_2, SaO_2 and Oxygen Content

The following section is adapted from Chapter 5 of Dr. Martin's book ***All You Really Need to Know to Interpret Arterial Blood Gases***, 2nd edition, published February 1999 by Lippincott Williams & Wilkins. Click on the title to see the Preface and Table of Contents. The book is available for purchase from the publisher at 1-800-638-0672 or 1-410-528-4223 and also at the following web sites:
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How much oxygen is in the blood? The Differences Between PaO_2, SaO_2 and Oxygen Content.

In the field of blood gas interpretation, confusion about PaO_2, SaO_2 and oxygen content is second only to confusion about mixed acid-base disturbances.

Arterial PO_2 (little 'a')gives us valuable information about adequacy of gas exchange within the lungs, when (and only when) it is subtracted from the calculated alveolar PO_2 (big A). We use the **Alveolar Gas Equation** to calculate PAO_2. The difference between measured PaO_2 and calculated PAO_2 is called the Alveolar-arterial PO2 difference or 'A-a Gradient' for short. The A-a gradient answers the important question: Are the lungs transferring oxygen properly from the atmosphere to the pulmonary circulation? If the A-a gradient is elevated, the answer is NO. If the A-a gradient is normal is YES. (The A-a gradient is discussed in detail in Chapter 4).

There is a second, equally important question concerning oxygen and gas exchange, which is the subject of this section:

How much oxygen is in the blood, and is it adequate for the patient?

The answer here must obviously be based on some oxygen value, but which one? After all, blood gases give us *three* different oxygen values:

- PaO_2
- SaO_2
- Oxygen content (CaO_2)

Of these three values, PaO_2, or *oxygen pressure*, is the least helpful to answer the question about oxygen adequacy in the blood. The other two values --*oxygen saturation* and *oxygen content* -- are more useful for this purpose. I will briefly define these three terms and then present a more detailed discussion of each, with emphasis on their inter-relationships.

OXYGEN PRESSURE: PaO_2.

Oxygen molecules dissolved in plasma (i.e., not bound to hemoglobin) are free to impinge on the measuring oxygen electrode. This "impingement" of free O_2 molecules is reflected as the partial pressure of oxygen; if the sample being tested is arterial blood, then it is the PaO_2. Although the number of O_2 molecules dissolved in plasma determines, along with other factors, how many

molecules will bind to hemoglobin, once bound the oxygen molecules *no longer exert any pressure* (bound oxygen molecules are no longer free to impinge on the measuring electrode). Since PaO_2 reflects only free oxygen molecules dissolved in plasma and not those bound to hemoglobin, PaO_2 cannot tell us "how much" oxygen is in the blood; for that you need to know how much oxygen is also bound to hemoglobin, information given by the SaO_2 and hemoglobin content.

OXYGEN SATURATION: SaO_2.

Binding sites for oxygen are the heme groups, the Fe^{++}-porphyrin portions of the hemoglobin molecule. There are four heme sites, and hence four oxygen binding sites, per hemoglobin molecule. Heme sites occupied by oxygen molecules are said to be "saturated" with oxygen. The percentage of all the available heme binding sites saturated with oxygen is the hemoglobin oxygen saturation (in arterial blood, the SaO_2). Note that SaO_2 alone doesn't reveal how much oxygen is in the blood; for that we also need to know the hemoglobin content.

OXYGEN CONTENT: CaO_2.

Tissues need a requisite amount of O_2 molecules for metabolism. Neither the PaO_2 nor the SaO_2 provide information on the number of oxygen molecules, i.e., of *how much* oxygen is in the blood. (Note that neither PaO_2 nor SaO_2 have units that denote any quantity.) Of the three values used for assessing blood oxygen levels, *how much* is provided only by the oxygen content, CaO_2 (units ml O_2/dl). This is because CaO_2 is the only value that incorporates the hemoglobin content. Oxygen content can be measured directly or calculated by the oxygen content equation (introduced in Chapter 2):

$$CaO_2 = Hb\ (gm/dl) \times 1.34\ ml\ O_2/gm\ Hb \times SaO_2 + PaO_2 \times (.003\ ml\ O_2/mm\ Hg/dl).$$

* * *

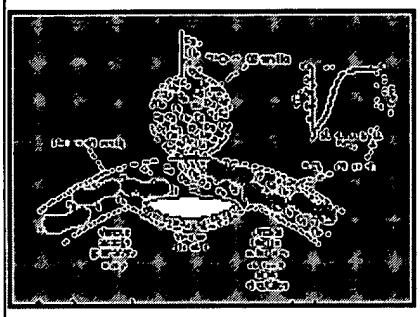
More on the definitions and distinctions of PaO_2, SaO_2 and CaO_2

You wish it was this simple, huh? I have shown the 3 short paragraphs above to dozens of students, interns, residents; almost all will say they understand the differences, no problem. But, when given questions to test their understanding, they flub. So more instruction is needed (and, yes, a few problems along the way). Understanding the differences between PaO_2, SaO_2 and CaO_2 is essential to proper blood gas interpretation. By the end of this and the next chapter -- if you work on all the problems -- you should be able to teach the subject!

PaO_2, the partial pressure of oxygen in the plasma phase of arterial blood, is registered by an electrode that senses randomly-moving, dissolved oxygen molecules. The amount of dissolved oxygen in the plasma phase -- and hence the PaO_2 -- is determined by alveolar PO_2 and lung architecture only, and is unrelated to anything about hemoglobin. (With one exception: when there is both anemia *and* a sizable right to left shunt of blood through the lungs. In this situation a sufficient amount of blood with low venous O_2 content can enter the arterial circulation and lead to a reduced PaO_2. However, with a normal amount of shunting, anemia and hemoglobin variables *do not* affect PaO_2.)

Oxygen molecules that pass through the thin alveolar-capillary membrane enter the plasma phase as dissolved (free) molecules; most of these molecules quickly enter the red blood cell and bind with hemoglobin (Figure 5-1). There is a dynamic equilibrium between the freely dissolved and the hemoglobin-bound oxygen molecules. However, the more dissolved molecules there are (i.e., the greater the PaO_2) the more will bind to available hemoglobin; thus SaO_2 always depends, to a large degree, on the concentration of dissolved oxygen molecules (i.e., on the PaO_2).

Figure 5-1. Oxygen pressure, saturation and content. Schematic shows cross section of lungs and pulmonary circulation. (CO_2, nitrogen and other gas molecules are omitted for clarity.) PaO_2 is always slightly lower than PAO_2 because of normal venous admixture, here represented by a connection between the venous and pulmonary circulations. See text for discussion. Click on figure to obtain larger image.



In this figure:

Hemoglobin content = 15 gm/dl
Alveolar partial pressure of oxygen (PAO_2) = 102 mm Hg
Venous partial pressure of oxygen (PvO_2) = 40 mm Hg
Venous hemoglobin oxygen saturation (SvO_2) = 75%
Arterial partial pressure of oxygen (PaO_2) = 95 mm Hg
Arterial hemoglobin oxygen saturation (SaO_2) = 97%

Because there is a virtually unlimited supply of oxygen molecules in the atmosphere, the dissolved O_2 molecules that leave the plasma to bind with hemoglobin are quickly replaced by others; once bound, oxygen no longer exerts a gas pressure. Thus hemoglobin is like an efficient sponge that soaks up oxygen so more can enter the blood. Hemoglobin continues to soak up oxygen molecules until it becomes saturated with the maximum amount it can hold - an amount that is largely determined by the PaO_2. Of course this whole process is near instantaneous and dynamic; at any given moment a given O_2 molecule could be bound or dissolved. However, depending on the PaO_2 and other factors, a certain percentage of all O_2 molecules will be dissolved and a certain percentage will be bound (Figure 5-1). In Figure 5-1, the free or dissolved oxygen molecules register a partial pressure of 95 mm Hg and the red blood cells contain a total hemoglobin content of 15 gm/dl.

Each hemoglobin molecule has four Fe^{++}heme sites for binding oxygen. If there is no interference (as from carbon monoxide, for example), the free O_2 molecules bind to these sites with great avidity. The total *percentage* of sites actually bound with O_2 is constant for a given set of conditions, and is the 'saturation of blood with oxygen'. This is called SvO_2 and SaO_2 in the venous and arterial circulations, respectively; in Figure 5-1, the respective values are 75% and 97%. An SaO_2 of 97%